Original Article

A comparison of the prevalence of gastric ulceration in feral and domesticated horses in the UK

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Keywords: horse; EGUS; stomach; squamous; glandular

Summary

Equine gastric ulcer syndrome is common in domesticated horses, especially those performing in athletic endeavours. However, the prevalence of equine gastric ulcer syndrome in feral populations of horses is not documented. This study investigated the prevalence and severity of squamous and glandular gastric ulceration in an abattoir population of horses in the UK. Both squamous and glandular ulceration were more prevalent in domesticated horses when compared to the feral horses studied.

Introduction

Equine gastric ulcer syndrome (EGUS) has been widely investigated during the past decades due to its high prevalence in populations of domesticated horses. In the mature horse, the occurrence of gastric lesions has predominantly been reported in the squamous region of the equine stomach. Prevalence of squamous gastric lesions has been reported to range from 11% in leisure horses to >80% in competing racehorses and high level endurance horses (Murray *et al.* 1996; Vatistas *et al.* 1999; Begg and O'Sullivan 2003; Chameroy *et al.* 2006; Tamzali *et al.* 2011). Lesions found within the glandular area of the stomach have been reported less frequently, with prevalences of 17–33%, 54–64% and 47–65% reported in endurance horses, leisure/sports horses and Thoroughbred racehorses, respectively (Begg and O'Sullivan 2003; Nieto *et al.* 2004; Tamzali *et al.* 2011; Hepburn 2014; Sykes *et al.* 2015b). However, to the authors' knowledge, the prevalence of EGUS in feral horses has not been reported. The present study therefore aimed to compare the prevalence of EGUS in domestic and feral horses presenting to an abattoir for slaughter.

Materials and methods

Ethics

This study was performed in accordance with the relevant Home Office guidelines in the UK.

Horses

Horses were randomly selected during two separate visits to a certified abattoir in the UK between June and August. Horses were classified, as assessed by an abattoir employee, as feral on the basis that they were free ranging with little or no human interaction, and classified as domesticated on the basis that they had been under human care for a designated purpose. The feral population originated from the Dartmoor and Exmoor regions of the UK. History of the horse such as age, sex, breed or discipline of the domesticated horses was not available.

Data collection

Once classified as feral or domesticated, horses were slaughtered in accordance with appropriate UK legislation. Following slaughter, the gastrointestinal tracts were removed and the stomachs separated by an abattoir employee. Each stomach was dissected along the dorsal plane from the lower oesophageal sphincter to the pyloric sphincter and rinsed with water. Stomachs were numbered and photographed for future analysis. Grading of the gastric lesions was performed by a single investigator (B.W.S.), blinded to the group allocation of the horse. Lesions were graded using the 4 point EGUS council system (Andrews *et al.* 1999) (**Table 1**) with the squamous and glandular regions of the stomachs graded separately.

Statistical analysis

Horses were dichotomously classified as being positive or negative for equine squamous gastric disease (ESGD) or equine glandular gastric disease (EGGD) if the assigned grade for the squamous or glandular mucosa was ≥II/IV or ≤I/IV, respectively (Sykes and Jokisalo 2015). A Chi-squared test was used to compare the prevalence of ESGD and EGGD between feral and domesticated horses, with a significance level of $P \leq 0.05$.

Results

A total of 60 domesticated horses and 29 feral horses were included in the study. Data from 2 feral and 9 domesticated horses were excluded due to inconclusive photographic documentation, resulting in 51 domesticated and 27 feral horses being included in the final analysis. **Figures 1** and **2** demonstrate the lesion distribution of ESGD and EGGD. Using the dichotomous classification, ESGD was present in 60.8% of domestic horses and 22.2% of feral horses ($P = 0.001$) and EGGD was present in 70.6% of domestic horses and 29.6% of feral horses ($P \leq 0.001$). ESGD and EGGD were present simultaneously in 42.3% and 7.4% of domestic and feral horses, respectively ($P \leq 0.001$).

Discussion

To the authors' knowledge, this is the first study to investigate the occurrence of EGUS in feral horses. The findings of the

TABLE 1: Grading system used for the classification of lesions (adapted from the EGUS council grading system).

Grade	Definition
0/IV	No lesions present
I/IV	Hyperaemia/hyperkeratosis and/or mild surface erosions, but no ulcerations
II/IV	Single large or several smaller superficial lesions
III/IV	Single layer/deeper lesions or multiple lesions
IV/IV	Severe ulceration, large/deep, coalescing

current study demonstrate that the EGUS may be present in feral horses, albeit at a much lower prevalence than in domestic horses. The results of the current study further demonstrate that the prevalence of ESGD and EGGD is significantly higher in domesticated horses compared to feral horses. It has recently been highlighted that ESGD and EGGD should be considered as different disease entities (Sykes *et al.* 2015a). As such, they will be considered separately in the following discussion.

The prevalence of ESGD in the group of domestic horses within this study was comparable to previous studies (Luthersson *et al.* 2009; Hepburn 2014), which obtained their results through endoscopic examination. Fundamentally, the pathophysiology of ESGD has been linked to the exposure of the *stratum corneum* of the squamous mucosa, a tissue with minimal protective mechanisms, to hydrochloric acid, secreted from the glandular area of the stomach, and volatile fatty acids, produced as a result of fermentation of soluble carbohydrates (Nadeau *et al.* 2003; Andrews *et al.* 2006) with bile acids potentially further exacerbating the risk of disease (Berschneider *et al.* 1999). Exercise further increases the risk of disease by pushing acidic gastric contents up onto the squamous mucosa due to the increased intra-abdominal pressure that is associated with gaits faster than a walk (Lorenzo-Figueras and Merritt 2002). These factors are particularly associated with the domestication of horses and probably explain the high prevalence of ESGD observed in the current study. Likewise, the absence of such risk factors is a logical explanation for the relatively low prevalence of ESGD observed in the feral horse population. The presence of ESGD lesions in 22.2% of feral horses was higher than expected by the authors. However, it has been previously shown that severe ESGD may develop rapidly with fasting (Murray and Eichorn 1996) and transport (McClure *et al.* 2005), to both of which the feral horses were exposed following capture and prior to slaughter and which may have contributed to the disease observed in this population.

Interestingly, the prevalence of EGGD in domesticated horses reported in this study (70.6%) was higher than previously reported in any other population (17–33%, 54–64% and 47–65% in endurance horses, leisure/sports horses and Thoroughbred racehorses, respectively) (Begg and O'Sullivan 2003; Nieto *et al.* 2004; Tamzali *et al.* 2011; Hepburn 2014; Sykes *et al.* 2015b). One possible explanation for this is that endoscopic examinations may be lacking in sensitivity when determining ulcerations within the glandular area of the stomach, particularly low grade lesions, when compared to necropsy (Andrews *et al.* 2002). However, it must be taken into account that the classification of the ulcerations in the present study was performed from photographic evidence rather than through histopathological examinations, which has been suggested to improve accuracy of diagnosis (Andrews *et al.* 2002). As such, the higher prevalence observed may be truly reflective of a higher rate of disease in the abattoir population than other populations previously studied.

At present, the pathophysiology EGGD is not known, although it is believed to result from a breakdown in normal mucosal defence mechanisms (Sykes *et al.* 2015a). The factors that contribute to this are yet to be elucidated but the finding of a significantly higher prevalence in domestic horses (70.6%) than feral horses (29.6%) suggests that intensive management may at least play a role in increasing the risk and severity of disease. This is consistent with a finding in endurance horses where the prevalence of EGGD increased from 17% during the interseason period to 33% within the season (Tamzali *et al.* 2011). The lack of a history on

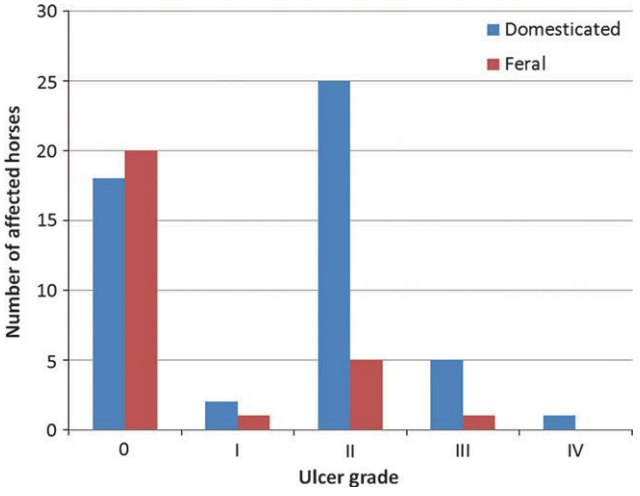

Fig 1: Equine squamous gastric disease lesion distribution in domesticated (n = 51) and feral (n = 27) horses presenting to an UK abattoir for slaughter.

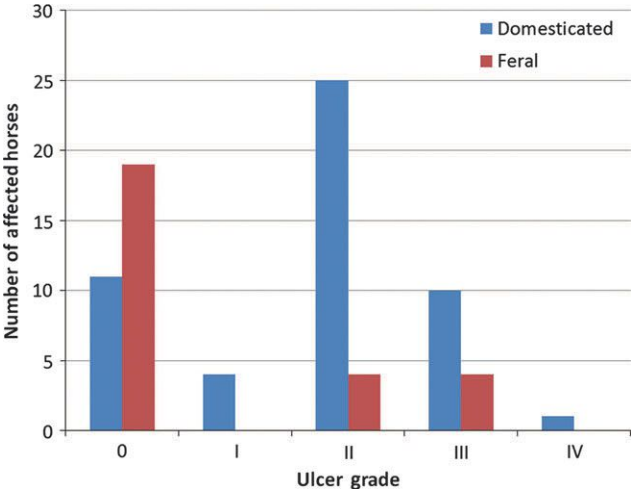

Fig 2: Equine glandular gastric disease lesion distribution in domesticated (n = 51) and feral (n = 27) horses presenting to an UK abattoir for slaughter.

management factors relating to the domestic horse population in this study makes further discussion as to the potential reasons for this finding purely speculative. Similar to ESGD, the finding of an EGGD prevalence of 29.6% in the feral horse population was surprising to the authors. Whether factors such as fasting and transport were associated with the development of disease in this population, as discussed above for ESGD, is not known but warrants consideration. Alternatively, EGGD may be present at a certain background prevalence within the feral horse population. Further studies investigating risk factors for EGGD and the prevalence of disease in feral populations not subject to capture, transport and holding are warranted.

Conclusions

The findings of the current study demonstrate that the prevalences of both ESGD and EGGD are higher in domesticated horses compared to feral horses, and that both are present within an abattoir population of feral horses. Further studies into the prevalence of disease in feral horse populations and the risk factors for EGGD are warranted.

Authors' declaration of interests

No conflicts of interest have been declared.

Ethical animal research

This study was performed in accordance with the relevant Home Office guidelines in the UK.

Source of funding

None.

Authorship

S. Ward contributed to study design and execution. B. Sykes contributed to study execution, data analysis and interpretation. H. Brown, A. Bishop and L.A. Penaluna contributed to study design. All authors contributed to preparation of the manuscript and approval of the manuscript.

References

Andrews, F., Bernard, W., Byars, D., Cohen, N., Divers, T., MacAllister, C., McGladdery, A., Merritt, A., Murray, M., Orsini, J., Snyder, J. and Vatistas, N. (1999) Recommendations for the diagnosis and treatment of equine gastric ulcer syndrome (EGUS). *Equine Vet. Educ.* **11**, 262-272.

Andrews, F.M., Reinemeyer, C.R., McCracken, M.D., Blackford, J.T., Nadeau, J.A., Saabye, L., Sotell, M. and Saxton, A. (2002) Comparison of endoscopic, necropsy and histology scoring of equine gastric ulcers. *Equine Vet. J.* **34**, 475-478.

Andrews, F.M., Buchanan, B.R., Smith, S.H., Elliott, S.B. and Saxton, A.M. (2006) *In vitro* effects of hydrochloric acid and various concentrations of acetic, propionic, butyric, or valeric acids on bioelectric properties of equine gastric squamous mucosa. *Am. J. Vet. Res.* **67**, 1873-1882.

Begg, L.M. and O'Sullivan, C.B. (2003) The prevalence and distribution of gastric ulceration in 345 racehorses. *Aust. Vet. J.* **81**, 199-201.

Berschneider, H.M., Blikslager, A.T. and Roberts, M.C. (1999) Role of duodenal reflux in nonglandular gastric ulcer disease of the mature horse. *Equine Vet. J.* **31**, *Suppl.* **29**, 24-29.

Chameroy, K.A., Nadeau, J.A., Bushmich, S.L., Dinger, J.E., Hoagland, T.A. and Saxton, A.M. (2006) Prevalence of non-glandular gastric ulcers in horses involved in a university riding program. *J. Equine. Vet. Sci.* **26**, 207-211.

Hepburn, R.J. (2014) Endoscopic examination of the squamous and glandular gastric mucosa in sport and leisure horses: 684 horses (2005–2011) [abstract]. In: *Proceedings of the 11th International Equine Colic Research Symposium*. p 5.

Lorenzo-Figueras, M. and Merritt, A. (2002) Effects of exercise on gastric volume and pH in the proximal portion of the stomach of horses. *Am. J. Vet. Res.* **63**, 1481-1487.

Luthersson, N., Nielsen, K.H., Harris, P. and Parkin, T.D.H. (2009) The prevalence and anatomical distribution of equine gastric ulceration syndrome (EGUS) in 201 horses in Denmark. *Equine Vet. J.* **41**, 619-624.

McClure, S., Carithers, D. and Gross, S. (2005) Gastric ulcer development in horses in a simulated show or training environment. *J. Am. Vet. Med. Ass.* **227**, 775-777.

Murray, M.J. and Eichorn, E.S. (1996) Effects of intermittent feed deprivation, intermittent feed deprivation with ranitidine administration, and stall confinement with *ad libitum* access to hay on gastric ulceration in horses. *Am. J. Vet. Res.* **57**, 1599-1603.

Murray, M.J., Schusser, G.R.F., Pipers, F.S. and Gross, S.J. (1996) Factors associated with gastric lesions in Thoroughbred racehorses. *Equine Vet. J.* **28**, 368-374.

Nadeau, J.A., Andrews, F.M., Patton, C.S., Argenzio, R.A., Mathew, A.G. and Saxton, A.M. (2003) Effects of hydrochloric, valeric, and other volatile fatty acids on pathogenesis of ulcers in the nonglandular portion of the stomach of horses. *Am. J. Vet. Res.* **64**, 413-417.

Nieto, J.E., Snyder, J.R., Beldomenico, P., Aleman, M., Kerr, J.W. and Spier, S.J. (2004) Prevalence of gastric ulcers in endurance horses – a preliminary report. *Vet. J.* **167**, 33-37.

Sykes, B.W. and Jokisalo, J.M. (2015) Rethinking equine gastric ulcer syndrome: Part 2 – equine squamous gastric ulcer syndrome (ESGUS). *Equine Vet. Educ.* **27**, 264-268.

Sykes, B.W., Hewetson, M., Hepburn, R.J., Luthersson, N. and Tamzali, Y. (2015a) European College of Equine Internal Medicine – Consensus Statement equine gastric ulcer syndrome (EGUS) in adult horses. *J. Vet. Intern. Med.* **29**, 1288-1299.

Sykes, B.W., Sykes, K.M. and Hallowell, G.D. (2015b) A comparison of three doses of omeprazole in the treatment of gastric ulceration in the horse: a randomised, blinded clinical trial. *Equine Vet. J.* **47**, 285-290.

Tamzali, Y., Marguet, C., Priymenko, N. and Lyazrhi, F. (2011) Prevalence of gastric ulcer syndrome in high-level endurance horses. *Equine Vet. J.* **43**, 141-144.

Vatistas, N.J., Snyder, J.R., Carlson, G., Johnson, B., Arthur, R.M., Thurmond, M., Zhou, H. and Lloyd, K.L.K. (1999) Cross-sectional study of gastric ulcers of the squamous mucosa in Thoroughbred racehorses. *Equine Vet. J.* **31**, 34-39.